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Measurement of Current Exposure to Environmental Tobacco Smoke

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ABSTRACT. Reports of recent exposure to environmental tobacco smoke (ETS) and urinary cotinine levels were obtained on 663 never- and ex-smokers who attended a cancer screening clinic in Buffalo, New York, in 1986. Study objectives included determining the prevalence of exposure to ETS using urinary cotinine and identifying questionnaire exposure measures predictive of cotinine. Findings demonstrate that exposure to environmental tobacco smoke is extremely prevalent, even among those not living with a smoker. A total of 76% of subjects reported exposure to ETS in the 4 d preceding the interview. The most frequently mentioned sources of exposure were at work (28%) and at home (27%). Cotinine was found in the urine of 91% of subjects. Cotinine values increased significantly with the number of exposures reported. Among the different questionnaire measures of exposure that were evaluated, the single best predictor of cotinine was the number of friends and family members seen regularly by the subject who smoke.

EXPOSURE to environmental tobacco smoke (ETS) has been implicated as a cause of many adverse health consequences in nonsmokers.[1-3] Although the health risks associated with ETS are probably small in comparison to active smoking, given the high prevalence of smoking in the United States,[4] exposure to ETS is likely to be common, and the number of people adversely affected could be substantial.

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Health risks associated with ETS vary with exposure. Biological markers of tobacco smoke exposure have recently been used in an attempt to measure smoke absorption in nonsmokers.[1,2,5-10] Of the various biochemical markers of tobacco smoke exposure, there is general agreement that cotinine is the best marker. Cotinine is a metabolite of nicotine and is therefore specific to tobacco smoke. Cotinine is considered a better marker of exposure than nicotine because its longer half-life means it measures exposure over several days rather than hours.[6-10] The degree of exposure, as measured by cotinine, is likely to depend upon several environmental factors, including number of exposures, duration of exposure, intensity of exposure (i.e., number of smokers), and room ventilation characteristics.[1,10]

This study examines the relative importance of these environmental factors in predicting current ETS exposure, as measured by urinary cotinine, in a group of 663 non-smokers. In addition, information is presented on the prevalence and sources of exposure to tobacco smoke. The findings from this study should be useful to those interested in developing valid questionnaire measures of recent exposure to ETS and to health officials charged with assessing the need for regulations that restrict smoking in public places.

Materials and methods

The study population included adult men and women who attended the Roswell Park Memorial Institute Cancer Screening Clinic for a free cancer checkup during 1986. Screening exams were performed by a nurse practitioner or physician. Following the examination, clinic attendees were asked by the examiner if they wished to participate in a study on ETS. Subjects were informed that participation in the study would require a 30- to 40-min interview, provision of a urine sample, and a lung function test. Among those approached, about 70% volunteered to participate. The main reason given for refusal to participate was lack of time. Those willing to participate were directed into a private office where an interviewer provided a further description of the study and obtained a signed consent.

Interviewing began in February 1986 and ended in December 1986. Smokers and non-smokers were enrolled in the study up until July 1986, after which time only non-smokers were recruited. A total of 860 individuals were enrolled in the study. These included 380 never-smokers, 350 ex-smokers, and 130 current smokers. Subjects were classified as ex-smokers if they smoked at least one cigarette/pipe/cigar a day for ≥ 1 yr and had not used tobacco for at least 1 mo prior to the interview. Current smokers were classified as those subjects who currently smoked any quantity of tobacco. The data presented in this paper are restricted to never- and ex-smoker participants in the study ($N = 730$).

The age distribution of subjects was widespread, ranging from 18 to 84 yr (mean age = 54.7 yr). Ex-smokers were slightly older (56.8 yr) than never smokers (53.2 yr). A higher proportion of never smokers compared to ex-smokers were below age 40 yr (21% vs. 12%). Overall, 44% of subjects were male, and 90% were white. A significantly higher proportion of ex-smokers were male compared to never-smokers (55% vs. 34%). The majority of subjects were married (69%). Slightly more than one-third of never smokers were college graduates, in contrast to 25% among ex-smokers. Roughly half the study subjects were currently employed. In comparison to the adult population in Erie County, New York, the study sample over represented females and whites and under represented persons below 40 yr of age.

Data collection. Study subjects were interviewed in a private office by a trained interviewer. The interviewer questioned subjects about their current and past tobacco use habits, exposure to tobacco smoke at home and at work, and recent indoor exposure to tobacco smoke over a 4-d period preceding the interview. To aid recall, each of the 4 d was subdivided into three segments (i.e., morning, afternoon, evening), and subjects were asked the same questions for each portion of the day. For each portion of a day, subjects were asked to indicate whether they had been exposed indoors, not in a car, to smoke from an individual who was smoking. Those who answered "yes" were asked to report on the location and duration (measured in quarter hours) of exposure, the number of smokers present (within 10 ft), the size of each exposure location, and the air ventilation characteristics of each location (i.e., open windows, air conditioning). Subjects were asked to rate the size of each exposure location on a 3-point scale as follows: 1 = large, defined as auditorium size; 2 = medium, defined as kitchen or living room size; and 3 = small, defined as small, single-person office size.

For each portion of a day, subjects were also asked to indicate whether they had been exposed to one or more people smoking in a car. Those who answered "yes" were asked how many people were smoking, the duration of exposure (measured in quarter hours), and whether windows were open or air conditioning was being used. The location size score for exposures in a car were automatically coded a 3, which corresponded to a small indoor exposure location.

The recall interview was structured so that for each portion of a day the subject could report on a single indoor exposure and one exposure occurring in a car. Thus, the maximum number of exposure events that could be recorded in a given day was 6 (3 segments in a day × 2 exposures per segment), and 24 for the entire 4-d recall period. If multiple exposure locations were reported in the same portion of the day, the more extensive exposure was recorded. Very few subjects reported multiple exposure locations during the same portion of a given day. Thus, the exposure reports recorded represent a fairly complete picture of a subject's perception of exposure to tobacco smoke over the 4 d preceding the interview.

In addition to information on tobacco smoke exposure, subjects were questioned about their current and past health status, work history, and personal characteristics. All subjects were given a lung function test and asked to provide a 6-ml urine sample for determination of cotinine.

Urine specimens were frozen at −80°C until the cotinine assay was performed. Assays were done within 6 mo of collection and without knowledge of the subject's smoking or exposure status. Cotinine was quantified using high pressure liquid chromatography (HPLC).[11] To check the accuracy of the assay, control samples with established mean cotinine values were performed with each HPLC run. If control values could not be repeated (i.e., coefficient of variation > 10%), the assay was redone.

Exposure measures. Several indicators of exposure to ETS were constructed from interview responses. From the information collected in the 4-d recall, five measures of ETS exposure were computed. These included (1) the total number of exposures, computed by summing the number of indoor and car exposures reported by subjects; (2) total duration of exposures, computed by summing across all reported exposures, the number of minutes exposed to tobacco smoke; (3) the intensity of exposures, measured by summing across all reported exposures, the number of smokers with whom the subject was exposed within 10 ft; (4) the size of exposure locations, computed by summing the size scores across all exposures; and (5) the ventilation characteristics of exposure locations measured by having subjects indicate for each location whether it was ventilated (i.e., open windows or air conditioning) or not (ventilated = 1, not ventilated = 0), and then summing the ventilation scores across all exposures.

In addition to measures derived from the 4-d recall, several general indicators of current ETS were assessed. These included (1) the number of cigarette/cigar/pipe smokers living in the subject's home (coded as none, one, two, or more); (2) among married subjects, the smoking status of their spouse; (3) among currently employed subjects, exposure to tobacco smoke at work; and (4) a rating by subjects of the number of people they see regularly (i.e., friends, relatives, coworkers) who smoke (response categories were none/few, some, most/all).

Data analysis. Analyses were restricted to lifelong non-smokers and ex-smokers. Two subjects who used chewing tobacco were excluded from analyses. Other exclusions included 45 subjects from whom urine samples were not obtained or were lost; 14 subjects for whom the cotinine assay was judged to be unreliable, i.e., coefficient of variation greater than 10%; and 6 subjects whose cotinine levels exceeded 90 ng/ml and were, therefore, classified as active smokers. The cutpoint of 90 ng/ml to distinguish between active and passive smoking was based on a comparison of the distributions for reported non-smokers and current smokers. Subjects excluded from the analysis did not differ significantly from those retained in the analysis with regard to demographic characteristics or self-reports of exposure to ETS.

The bivariate relationship between urinary cotinine and measures of ETS smoke exposure were evaluated by either one-way analysis of variance or Pearson Product Moment correlation coefficients, as appropriate. Multiple regression analysis was employed to evaluate the relationship between urinary cotinine and measures of exposure to passive smoke, controlling for the following potential confounding variables: age; sex; time of day when the specimen was collected (coded as: morning, afternoon, evening); and time of year when the specimen was collected (coded as: indoor months = November through April and outdoor months = May through October).

One-way analysis of variance was used to evaluate the relationship between the characteristics of subjects and exposure to ETS as measured in the 4-d recall. A stepwise multiple regression analysis was performed to assess the multivariate importance of variables found in the bivariate analysis to be associated with reported exposures.

Results

Seventy-six per cent (501/663) of the subjects reported exposure to tobacco smoke in the 4 d preceding the interview. The average number of exposures reported over the 4-d period was 3.3 (range: 0 to 21 exposures). Among the 501 exposed subjects, the average daily exposure was 2 h (range < .1 h to 13.25 h/d). Reported exposure locations in order of frequency were work (28%), home (27%), restaurants (16%), private social gatherings (11%), in a car or airplane (10%), and in public buildings (8%).

Twenty-two per cent of subjects (n = 145) lived with a smoker. Of the 466 married subjects, 94 reported that their spouse smoked. Among currently employed subjects (n = 343), 77% reported being exposed to tobacco smoke at work. Twenty per cent of subjects stated that smoking is prohibited in their home; 40% prohibited smoking in their car. Fifty-seven per cent of subjects reported that none or few of their family and friends smoke, 29% said that some smoke, and 14% said that most or all smoke.

Six hundred and five of the 663 (91%) had detectable cotinine levels. The mean cotinine level was 8.84 ng/ml (median = 6.19 ng/ml). Cotinine levels ranged from 0 ng/ml to 85 ng/ml; 92% of cotinine values were less than 20 ng/ml.

Figure 1 shows the mean urinary cotinine levels by the number of exposures reported by subjects during the 4-d recall period. Whereas concentrations of cotinine varied widely within exposure groups, the level increased with the number of exposures reported (Pearson Product Moment Correlation = 0.23, $p < .01$).

Table 1 shows the relationship between urinary cotinine and various measures of exposure to ETS computed from the 4-d recall portion of the interview. All exposure measures were significantly related to cotinine, although the degree of association was modest. Exposure measures were highly intercorrelated because each was based on the number of exposure occurrences (range: r = 0.70 to r = 0.96). To evaluate the relationship of exposure duration, intensity, room size, and room ventilation with cotinine, independent of number of exposures, partial correlations were computed controlling for number of exposures. The partial correlation coefficients are shown in the second column in Table 1. When the number of exposure occurrences was controlled, only the ventilation character-

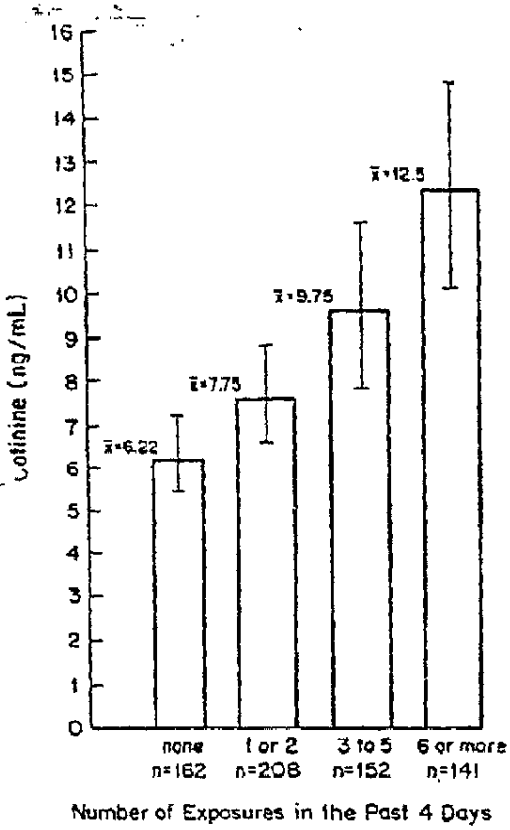

Fig. 1. Urinary cotinine concentrations by number of reported exposures to tobacco smoke in the past 4 d among 663 nonsmokers, Buffalo, New York, 1986.

istics of the exposure location was found to be significantly related to cotinine levels.

A multiple regression anlaysis was performed to further evaluate the association between the number of exposure reports and cotinine while controlling for the age and sex of the subject and the time of day and month of year when the urine specimens were collected. Overall, this model accounted for 8% of the variability in cotinine levels. Number of exposures and the time of year when urine specimens were collected were the only variables significantly related to cotinine. Each exposure occurrence increased cotinine by 0.58 ng/ml (95% confidence interval: 0.36 ng/ml to 0.80 ng/ml).

Cotinine levels were significantly higher in subjects interviewed during cold weather months (November to April), in subjects who lived with smokers, and in those who reported that most or all of their friends and family whom they see regularly smoke. Cotinine levels also varied significantly by age and race. Younger subjects and nonwhites had higher cotinine levels.

A stepwise multiple regression analysis was performed to evaluate the predictive value of variables found in the bivariate analysis to be associated ($p < .10$) with cotinine. Variables included in the regression model were the subject's age, race, and employment status; the number of smokers who lived with the subject; the number of friends and family members seen regularly who smoked; and the time of the year when the interview was conducted. These six variables accounted for only 7% of the variability in cotinine levels. Three of the six variables were significant contributors to the model. These included, in order of importance, number of friends and family members who smoked; the time of the year when the interview was conducted (cotinine levels higher in subjects interviewed during indoor months, November to April); and the number of smokers who lived with the subject.

The relationship between the characteristics of subjects and reported exposure to passive smoke as measured in the 4-d recall was also assessed. Age, living with a smoker, number of friends and family members who smoked; rules governing smoking at home and in the car, working in a place where smoking is allowed, and time of year when the interview was conducted were all significantly associated with the number of reported exposures. A stepwise multiple regression analysis was performed to evaluate the predictive value of variables found in the bivariate analysis to be associated ($p < .10$) with number of exposures. Variables included in the regression model were the subject's age, employment status, number of smokers who lived with the subject, rules governing smoking at home and in the car, number of friends and family members seen regularly by the subject who smoked, and month of the year when the interview was conducted. These seven variables accounted for 35% of the variance in reported exposure to passive smoke. Six of the seven variables were significant contributors in the model. These included, in order of importance, the number of smokers who lived with the subject; the number of friends and family members seen regularly by the subject who smoked; the subject's employment status (more exposures reported by those currently employed); rules governing smoking in the subject's car;

Table 1.—Relationship between Cotinine (ng/ml) and Measures of Exposure to Passive Smoke from the 4-d Recall

Exposure measures	Correlation with cotinine (ng/ml)	
	Unadjusted	Adjusted for number of exposures
Number of exposures	0.23*	—
Duration of exposure	0.18*	−0.02
Number of smokers	0.19*	0.01
Room size score	0.24*	0.05
Ventilation score	0.25*	0.09†

*$p < .05$.
†$p < .01$.

age (more exposures reported by younger subjects); and time of the year when the interview was conducted (more exposures reported by those interviewed during indoor months, November to April). Rules governing smoking at home was correlated with rules about smoking in the car ($r = 0.46$, $p < .01$), which may account for its failure to enter the model as a significant predictor of exposures.

Discussion

Given the self-selected nature of the study population and potentially limited generalizability of results, it is worth noting that the ETS exposure rates reported by study subjects are comparable with exposure rates reported in the literature.[13] Friedman et al.[13] found that, among 37 000 nonsmoking members of a prepaid medical plan who were questioned about their exposure to ETS, 63% indicated exposure to tobacco smoke in the previous week. Reported ETS exposure was strongly related to age, with adults in their twenties reporting the highest level of exposure. In this study, three-fourths of non-smokers interviewed reported exposure to tobacco smoke in the 4 d preceding the interview. Similar to the Friedman et al.[13] finding, ETS exposure was highest among respondents in their twenties and declined steadily with age.

The two most frequently mentioned locations for exposure to passive smoke were at work and at home. Among currently employed subjects, 77% reported being exposed to tobacco smoke at work. Over half of all reported recent exposures occurred in locations where the subject may not have the option to avoid exposure (i.e., at work, in a restaurant, in a public building). This finding suggests that policies regulating smoking in public places could have a substantial impact on reducing a person's exposure to ETS.

The mean urinary cotinine level of 8.84 ng/ml found among nonsmokers in this study is comparable to reports from other studies.[5,7] By contrast, the mean urinary cotinine level for the 130 smokers tested in this study was 1 254 ng/ml. Among nonsmokers, detectable levels of cotinine were found in the urine of 91% of subjects, including 132 of 162 subjects (81%) who reported no exposure in the 4 d preceding the interview. It is possible that cotinine levels were influenced by exposures that occurred earlier than 4 d reported on in the interview.[12] Also, it is our impression that subjects who are not routinely exposed to ETS may have difficulty recalling instances of exposure.

This study examined several self-reported environmental factors that may influence cotinine levels, including the number of exposures; duration; intensity, i.e., number of smokers; room size; and ventilation characteristics of exposure locations. Consistent with other published reports,[5-9] cotinine levels tended to increase with the number of reported exposures to ETS. However, within a given exposure level, there was considerable variability in cotinine values.

Cotinine was chosen as a biological marker of ETS exposure because it is specific to tobacco smoke. However, cotinine levels in body fluids may not only reflect environmental exposure to tobacco smoke, but also factors that influence uptake and metabolism of nicotine.[10-12] In controlled laboratory conditions (smoke chambers), it has been shown that duration and intensity of exposure to ETS can affect absorption of nicotine.[10] Results from this study show that accounting for exposure duration and intensity had little influence on cotinine levels once the number of exposures was controlled. Considering the room size and ventilation characteristics of the exposure location also added little to predicting variation in cotinine levels. In questionnaire studies of ETS, it does not appear to be useful to account for characteristics of exposure location, i.e., duration, number of smokers, room size, ventilation factors. Instead, more emphasis should be placed on frequency measures of exposure and the number of smokers among acquaintances.

Findings from this study confirm the results of other investigations, which have found that living with a smoker increases cotinine levels.[5-9] However, 84% of subjects who did not live with a smoker had detectable cotinine levels, which underscores the need to consider exposures outside the home. Among the various general exposure measures examined, the best predictor of cotinine was the number of friends and family members seen regularly by the subject who smoked. This measure considers home, workplace, and social exposures to tobacco smoke, and it represents a simple way to evaluate a nonsmoker's usual exposure to ETS.

Cotinine levels were found to vary by month of the year. Subjects who were interviewed during predominantly cold weather months (November to April) reported more frequent exposure to ETS and exhibited significantly higher cotinine levels than subjects interviewed during warm weather months (May to October). The time of the year may not only influence the number of exposures to ETS but also the ventilation characteristics of exposure locations.

Cotinine was assumed to be a valid quantitative measure of ETS exposure in this study. However, there were several potential problems with the cotinine values. Because of the way in which subjects were recruited, it was not possible to fix the day of the week or the time of day when specimens were collected. Jarvis et al.[6] found that plasma cotinine levels tend to increase in the afternoon. Time of day when the interview was conducted was examined as a potential confounding variable in this study and was found to be unrelated to cotinine levels. Another potential problem with the measurement of cotinine in this study is that values were based on a single random urine specimen. Preferably, cotinine levels should be based on 24-h urine collection to control for variability in the concentration of cotinine between individual urine specimens. In an effort to control for variability in urinary concentrations of cotinine, values were standardized by creatinine excretion, which served as a surrogate measure of urine concentration, and expressed as a cotinine:creatinine ratio. However, parallel analyses done on standardized and unstandardized cotinine values revealed that the correction for creatinine had little effect on the results.

A recent workshop on the measurement of cotinine in nonsmokers recommended, for comparison purposes across studies, that unstandardized values be presented,[14] which is why we have chosen to present our unstandardized cotinine levels.

The relatively modest correlation between reported ETS exposure and urinary cotinine indicates that other factors such as differing metabolic rates and body size may have a confounding effect on the relationship between cotinine levels and questionnaire measures of ETS exposure. In view of this finding, we would recommend against using cotinine levels as a strictly quantitative indicator of ETS. The combination of questionnaire measures of exposure and biologic markers offers perhaps the best approach for accurately assessing recent exposure to ETS.[15]

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